

REMARKS/ARGUMENTS

Claims 1, 3, 6 and 7 are pending in the application. Claim 1 has been amended. Applicant reserves the right to present any withdrawn or canceled subject matter in one or more continuation or divisional applications.

Rejections under 35 U.S.C. §103

Claims 1, 3, 6 and 7 have been rejected under 35 U.S.C. § 103(a) as obvious over U.S. Patent No. 5,212,158 to Vandai in view of Hock et al., U.S. Patent No. 5,043,346.

Vandai discloses derivatives of L-proline described as possessing a nootropic action, and methods of treating amnesia using the compounds. As noted by the Examiner, Vandai does not disclose treatment of a postlesional neuronal disease due to cerebral infarction or traumatic impact characterized by nerve cell necrosis.

Hock states that “a substance is designated as having nootropic activity when it is able to abolish the amnesia produced in the experimental animals by means of an electroconvulsive shock or the amnesia induced by scopolamine.” Col. 9, lines 11-16 of Hock.

Neither Vandai or Hock, alone or in combination, disclose or suggest treatment of a postlesional neuronal disease due to cerebral infarction or traumatic impact characterized by nerve cell necrosis, thereby effecting nerve cell regeneration, as recited in the amended claims. There is no suggestion from Vandai or Hock of the methods defined by the claims, in the absence of hindsight reconstruction.

Vandai does not disclose or suggest a mechanism of action of the compounds described therein that would indicate any suitability for use in postlesional diseases characterized by nerve cell necrosis, as recited in the claims. Vandai discloses the alleged *nootropic* effects of the described compounds.

The promotion of neurite growth – which is a prerequisite for the treatment of postlesional diseases of necrotic origin – does not belong to one of the possible mechanisms of action known from the literature, and has no connection with the mechanism of action known for nootropics.

The postlesional neuronal diseases recited in the claims are due to cerebral infarction or traumatic impact "characterized by nerve cell necrosis", that is, nerve cell death. In the methods of treatment claimed, it is required that the nerve cells be stimulated to grow. The present application demonstrates the effectiveness of the compounds recited in the claims for the treatment of specific neuronal diseases due to their efficacy in the stimulation of nerve growth. In the prior art, there was no recognition of this effect, and therefore, there would have been no motivation to treat the specific conditions recited in the claims. There would have been no motivation to administer the compounds recited in the claims to stimulate nerve growth and treat postlesional neuronal diseases due to cerebral infarction or traumatic impact characterized by nerve cell necrosis, in view of Vandai and Hock, since these references when combined fail to disclose or suggest stimulation of nerve growth, or the requirement thereof for the treatment of amnesia.

There is no basis in the references for the Examiner's statement in paragraph 3 of page 3 of the outstanding Office Action that necrotic cell death is a functional effect of amnesia induced by electroconvulsive shock or scopolamine. There is no suggestion in Vandai that nerve cell growth is required or occurs in the disclosed treatments of amnesia. In fact, Vandai merely discloses the treatment of chemically induced amnesia, with no suggestion that nerve growth is required or occurs.

The claimed methods for treating postlesional neuronal diseases due to cerebral infarction or traumatic impact characterized by nerve cell necrosis are thus non-obvious in view of Vandai and Hock. There is no suggestion of the use of the compounds recited in the claims to effect regenerative processes which are essential for the treatment of postlesional diseases of the nervous system due to cerebral infarction or traumatic impact characterized by nerve cell necrosis.

In the pertinent literature, a difference is drawn between a therapeutic treatment of neurodegenerative conditions, for which nootropics are used, such as for example Alzheimer's disease, and *regenerative* processes, which are necessary for the treatment of postlesional diseases of the nervous system, as presently claimed (see Varon and Connor (1994) "Nerve Growth Effector in CNS Repair" in *Journal of Neurotrauma*, vol. 11, no. 5, Exhibit B, attached with Applicant's Amendment dated April 15, 2005).

This reference supports the contention that the nootropic or even anti-neurodegenerative effect of certain substances does not render their effect on regenerative processes obvious. The present application contains experimental data proving the neuro-regenerative effect of the compound Cinnamoyl-GFPNH₂, which supports the neuro-regenerative effect of the compounds recited in the claims. There is no suggestion in Vandai or Hock that the compounds recited in the amended claims have a regenerative effect in promoting nerve growth. Thus, one skilled in the art would not have been lead to use these compounds in a method of treatment of postlesional diseases characterized by necrotic cell death, as recited in the amended claims, which require such regeneration. There is no suggestion within the references applied by the Examiner that the compounds would have a neuroregenerative effect or would be effective to treat the specific diseases recited in the claims.

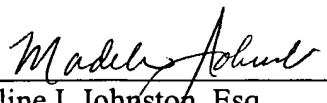
Vandai does not disclose or suggest the methods defined by the amended claims. Hock does not provide any additional teaching that would have suggested the claimed methods to one of ordinary skill in the art, in the absence of hindsight. Therefore withdrawal of this rejection is respectfully requested.

Conclusion

In view of the above arguments, withdrawal of the outstanding rejections is respectfully requested.

The Commissioner is authorized to charge any fees associated with this filing not attached hereto to Deposit Account 11-0980.

Respectfully submitted,

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